Acute Pancreatitis Complicating Hepatitis A - A Case Report

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Abstract

Acute pancreatitis complicating acute hepatitis A is very rare especially in children. We, hereby, report an 11 year old female patient with acute hepatitis A infection who later developed acute pancreatitis in course of the illness. She improved with conservative management and was discharged. This case highlights the association of pancreatitis with non-fulminant hepatitis which should be considered in endemic areas with acute abdomen.

Key Words: Hepatitis-A, Pancreatitis, Children

INTRODUCTION

Hepatitis A virus is an unusual cause of acute pancreatitis. The incidence of acute pancreatitis complicating acute viral hepatitis A infection is very low in children, though, it is mostly reported in young males and adolescents.[1,2] Although, hepatitis viruses have a strong affinity for hepatocytes, viral antigens have also been detected in other tissues such as the pancreas and gallbladder. Infectious cause was first documented by Lemoine in 1905 in a patient with mumps. Other viruses such as Coxsackie-B, Epstein-Barr virus, measles, influenza-A and varicella zoster have also been implicated in acute pancreatitis.[3] In 1944, Linsey first reported the association between acute pancreatitis and infectious hepatitis.[4] In fulminant hepatic failure (FHF), pancreatitis occurs in up to 34% of the cases; although, in non-fulminant hepatitis it is considered rare.[1,4] A recent pediatric series describes acute pancreatitis to be associated with hepatitis A in only 8.3% cases.[5] We report a case of an 11 year old girl with acute hepatitis A, who developed acute pancreatitis with pleural effusion in the course but had uneventful recovery.

CASE REPORT

An 11-year-old female child admitted with epigastric pain, nausea, anorexia, jaundice and fever of 4 days duration. The abdominal discomfort gradually increased in severity and was associated with vomiting. The pain was boring in nature, persisting throughout the day with the child lying curled up on her side in bed and feeling better when sitting up and stooping forward. She had no previous similar episodes, no abdominal trauma, diarrohea, parotid swelling, vaccination, blood transfusion, hepatic disease, gallstone, family history of pancreatitis or hyperlipidemia, recent travel, drug abuse, alcoholism or surgery.

Examination revealed jaundice and epigastric region tenderness during superficial palpation, with no signs of peritonitis. Her liver was enlarged, exceeding costal margin by 4 cm. Laboratory investigations showed: Hb 12.2 g/dL, and total leukocyte count of 10,800/uL, (total neutrophils 84%, lymphocytes 14%, monocytes 2%), platelets 205,000/µL, total bilirubin 4.9 mg/dL with direct bilirubin of 4.6 mg/dL, serum aspartate aminotransferase (AST) 1051 IU/L, alanine aminotransferase (ALT) 948 IU/L, lipase 1680 IU/L, amylase 1092 IU/L, serum calcium 8.5 mg/dL, triglyceride 133 mg/dL, serum alkaline phosphatase 415 IU/L, gamma-glutamyl transferase (GGT) 153 IU/L, serum albumin 3.1 g/dL, prothrombin time 14 sec (INR: 1.08), erythrocyte sedimentation rate (ESR) 39 mm/h. The serology for Hepatitis B and C were negative, and IgM anti-HAV was positive. Abdominal ultrasonography revealed hepatomegaly with reduced echotexture, gall bladder edema, and minimal free fluid in pelvic cavity. Clinical diagnosis of pancreatitis was made and CECT upper abdomen was done which showed bulky pancreas with homogenous enhancement and peripancreatic stranding

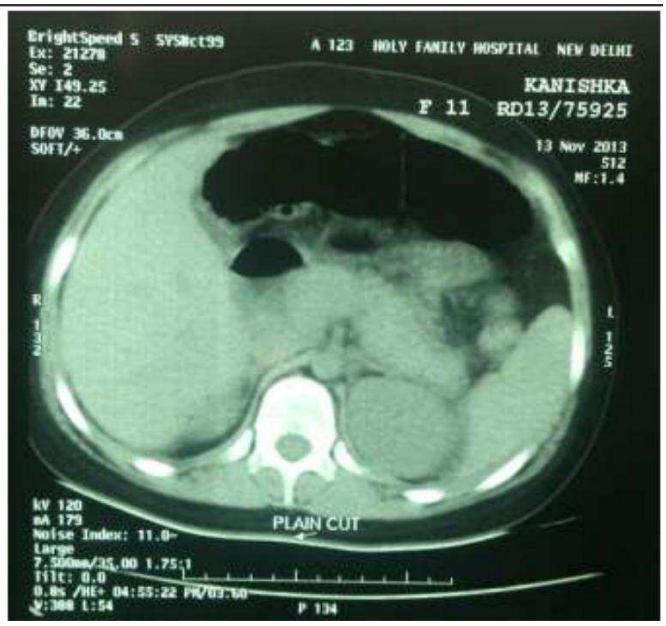

Figure 1 - CECT upper abdomen was done which showed area of necrosis (black arrow), overstretching of superior mesenteric artery (blue arrow), para-aortic lymph node (red arrow) and bulky pancreas with fuzzy margins (yellow arrow).

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with hepatomegaly and edematous gall bladder with minimal bilateral pleural effusion [Figure 1].

The patient received conservative management with analgesia, hydration and nil per oral. During follow-up, she remained stable, afebrile and her symptoms regressed within seven days. She was discharged on the 8th day of admission after significant clinical and laboratory improvement. During ambulatory follow-up the patient had complete resolution of the symptoms and biochemical results.

DISCUSSION

Acute hepatitis A is endemic in India. Acute pancreatitis has been reported very rarely in acute viral hepatitis. Few case reports and case series have documented such association.[1,4] The mechanism of pancreatitis in patients with acute viral hepatitis (non-fulminant) is unknown, and it may be multifactorial. A hypothesized pathogenesis of pancreatitis associated with hepatitis is the development of edema of the Ampulla of Vater and pancreatic duct leading to pancreatitis as result of obstruction to the outflow of pancreatic fluid.[6] There is no documented evidence of route of spread of hepatitis virus to the pancreas; however, the proposed routes are blood or bile.[7] The presence of hepatitis viral antigens on the surface and in the cytoplasm of the acinar cells of the pancreas supports the theory of inflammation and direct injury of the acinar cells by hepatitis virus. It is possible that the severity of pancreatitis is related to the magnitude of exposure of pancreatic acinar cells to the hepatitis virus.[8]

The hepatitis viruses might injure the pancreatic acinar cell membrane, resulting in the leakage of intracellular enzymes, and/or precipitate a network of intracellular events culminating in cell death by a mechanism analogous to hepatocyte necrosis.[9] Another mechanism can be the release and circulation of lysosomal enzymes from the inflamed liver with the activation of trypsinogen to trypsin. Hyperlipedemia in our case was taken as an association with acute pancreatitis rather than a precipating factor.

When acute pancreatitis is associated with fulminant hepatitis, the virus may cause tissue damage directly, but there are several other factors which can play an important role in the development of pancreatitis (clinical or silent) and these include acute liver failure, hypotension, infections and drug induced Damage.[10]

CONCLUSION

This case highlights the possibility of pancreatitis in viral hepatitis, which should be kept in mind in endemic areas whenever one is presented with features of acute abdomen. Conservative treatment leads to recovery in all patients.

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